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February 24., 2000

Honorable Carol M. Browner
United States Environmental
Protection Agency
401 M Street, S.W.
Room 1200, West Tower
Mail Code 1101
Washington, D.C. 20460

Re: Petition to Add Diisononyl Phthalate (DINP) to the Emergency Planning and Community Right-to-Know Act Section 313 List of Toxic Chemicals

Dear Administrator Browner:

We write on behalf of children and adults that are affected by production of DINP, a dangerous phthalate ester that is used as the principal plasticizer in toys and many other products used daily by children and adults. DINP has been shown to cause cancer and other very serious toxic effects. Most importantly, in every study conducted to measure DINP exposure from children's use of plastic, DINP has been shown to migrate from the plastic into saliva when the plastic object is chewed or put into the child's mouth (Babich, 1998). Governments worldwide will not allow its use in toys or other articles that children commonly use because it is so toxic and leachable. The United States Consumer Product Safety Commission (CPSC) has examined the health and safety data on DINP and determined that it is toxic and carcinogenic and requested that industry remove DINP from products intended for use by children under the age of three. Other governments have banned DINP.

Despite its known toxicity and widespread use in goods that the public freely uses on a daily basis, DINP is not listed on the Emergency Planning and Community Right-to-Know Act (EPCRA) Section 313 list of toxic chemicals. DINP must be listed, so that the public and local communities can exercise their right to have information relating to DINP that could directly and significantly affect their health and the health of their children. We urge you to add DINP to the toxic chemical list immediately, so that reporting on this dangerous chemical can begin.

What Is DINP

DINP is diisononyl phthalate, a member of a family of phthalic acid esters used as plasticizers in vinyl plastic to make it pliable. DINP is used as the principal plasticizer in toys, soft rattles, teethers, and some baby bottle nipples -- all products that children routinely put in their mouths. In addition, DINP is used in vinyl upholstery, wire and cable, and coated fabrics -- products that children and adults come into contact with on a daily basis (Babich, 1998 at 9). Moreover, DINP is found as a contaminant in food.

The actual composition of DINP varies with the manufacturing process and there are at least two current DINP manufacturers in the United States -- Exxon Chemical Corporation and Aristech Chemical Corporation. DINP is known by at least three Chemical Abstract Service (CAS) numbers -- 28553-12-0, 68515-48-0, and 71549-78-5. How the compositional differences in DINP affect toxicity is not known.

DINP Is Highly Toxic

DINP is highly toxic and very dangerous to our children and ourselves. DINP causes cancer, systemic toxicity, developmental toxicity, and endocrine disruption. DINP is more toxic than previously thought. Documents showing that DINP causes these effects are appended.

DINP Causes Cancer: Studies filed at EPA by one of DINP's manufacturers -- Aristech Chemical Corporation -- show unequivocally that DINP is a carcinogen. Aristech Chemical Corporation reported this to the EPA on three occasions in 1995 (Aristech, 1995a,b,c) and also reported this at scientific meetings (Butala *et al.*, 1996, 1997). The Aristech study reports show statistically significant cancers in DINP-treated male and female rats and mice. These cancers include liver carcinoma, leukemia, and kidney cancer. An earlier unpublished study (Bio/dynamics, 1986) predicted that DINP would be carcinogenic.

All three studies prevent reliance on the 1997 Lington study (Lington *et al.*, 1997), which finds that DINP dosed at less than 400 mg/kg/day is not carcinogenic. According to the CPSC report on chronic toxicity of DINP and children's products, "in the positive [for cancer] studies, increased [hepatocellular carcinoma] was consistently observed only at doses of at least 1 percent in the feed (about 600 mg/kg-d). Therefore, the earlier negative results (Lington *et al.*, 1997) may be explained by the selection of doses" (Babich, 1998 at 11). In fact, the animals in the Lington study did not reach a 10 percent level of body weight reduction in the study (the National Toxicology Program (NTP) rule of thumb for dose selection), and CPSC scientists concluded that the Lington study did not use adequate dose levels to produce a carcinogenic effect. It is likely that the animals in the Lington study would have developed cancers from DINP if higher doses had been used.

DINP Causes Systemic Toxicity: DINP produces toxic changes in the liver and kidneys. These toxic effects include spongiosis hepatis, liver cell enlargement, and mineralization of the renal papilla and pigments in renal tubule cells. DINP also causes blood abnormalities and metabolism abnormalities (Babich, 1998; Lee, 1998).

DINP Causes Developmental Toxicity: Metabolic breakdown products of DINP produce developmental retardation in fetuses at doses that have no effect on the pregnant animal (Lee, 1998). Scientists at the CPSC concluded that this "delayed development suggests a potentially adverse effect on the isononanol components of DINP" (Lee, 1998).

DINP Causes Endocrine Disruption: Data show that DINP is an endocrine disruptor. DINP has been found to be estrogenic in yeast cells and to stimulate human breast cancer cell division (Harris *et al.*, 1997).

DINP Causes Peroxisome Proliferation in the Liver, Which Can Lead to Cancer: DINP produces peroxisome proliferation in the liver. The literature -- which is reviewed in the CPSC Report on DINP -- establishes that peroxisome proliferating compounds can be carcinogenic, although the mechanistic link between peroxisome proliferation and cancer is unknown. A specific cellular receptor for peroxisome proliferation -- the PPAR -- has been identified in a number of animal species, including humans (Gonzalez, 1997). According to the CPSC Report, "[a]lthough humans express PPAR_ at a lower level than mice, the human PPAR_ was shown to function normally in mouse cells *in vitro*" (Babich, 1998 at 14). The CPSC Report goes on to state that peroxisome proliferating carcinogens, like DINP, are complete carcinogens and that "[s]cientists at the U.S. Environmental Protection Agency . . . and the National Institute for Environmental Health Sciences . . . regard [peroxisome proliferating carcinogen]-induced tumors as relevant to humans" (Babich, 1998 at 15).

DINP must be regarded as a cancer threat, even without the Aristech data, because it is a peroxisome proliferator. DINP is a complete carcinogen. The elements of the carcinogenic response demonstrated repeatedly in animal studies are present in humans. What is unknown is the level of susceptibility that humans, or different groups of humans, particularly children, will exhibit to this toxicity.

DINP Has Been Toxicologically Linked to DEHP, Which Causes Many Toxic Effects: Scientists at CPSC have compared the cancer potency of DINP to that of di(2-ethylhexyl) phthalate (DEHP), a related phthalate ester, and have concluded that "[t]he dose response [for cancer] observed with DINP is consistent with that of DEHP, another branched chain [dialkyl phthalate]" (Babich, 1998 at 11). DEHP, the most widely used and best studied phthalate ester, has been evaluated by the Agency for Toxic Substances and Disease Registry (ATSDR) (ATSDR, 1993), which concluded:

- In laboratory studies, short-term exposure to DEHP interfered with sperm function and caused decreased fertility. These effects were seen in two animal species -- rats and mice.

- In laboratory studies, DEHP produced fetal defects in two species -- rats and mice.

- These studies lead ATSDR to conclude that humans exposed to DEHP may have children with low birth weight and skeletal and/or nervous system defects.

- Long-term exposure of rats to DEHP has caused structural and functional changes

in the kidney. ATSDR states that the kidney structural changes in rats are similar to those seen in human patients on long-term dialysis -- patients that are exposed to DEHP as a result of their dialysis treatment.

Long-term exposure to DEHP in rats and mice causes cancer. As a result of these studies, the U.S. Public Health Service considers DEHP to be a carcinogen; the International Agency for Research on Cancer (IARC) classifies DEHP as a possible carcinogen; and EPA classifies DEHP as a probable human carcinogen.

DINP shares DEHP's toxicological characteristics. These conclusions reached by scientists at ATSDR for DEHP are equally applicable to DINP.

DINP Migrates From Plasticized PVC Toys, Pacifiers, And Teethers

The CPSC and the European Union have independently evaluated DINP migration from PVC toys and child-care articles as a result of chewing or mouthing and found that in every instance, DINP migrates from the plastic into saliva (Babich, 1998). The extent of DINP migration depends on the time the child spends chewing, but younger children, aged 3-12 months, received the highest doses of DINP -- higher than children aged 13-26 months. In this case, the most vulnerable members of society receive the greatest exposure to DINP.

Congress Intended Dangerous Chemicals Like DINP To Be Listed

A chemical like DINP that causes cancer and reproductive and developmental effects must be listed. Congress intended that exactly these types of dangerous chemicals be on the list, so that the public could have ready access to important information about them. The Section 313 listing standard is straight forward:

> A chemical may be added if the Administrator determines, in his judgment, that there is sufficient evidence to establish any one of the following:
>
> () The chemical is known to cause or can reasonably be anticipated to cause significant adverse acute human health effects at concentration levels that are reasonably likely to exist beyond facility site boundaries as a result of continuous or frequently recurring, releases.
>
> () The chemical is known to cause or can reasonably be anticipated to cause in humans --
>
> () cancer or teratogenic effects, or
>
> () serious or irreversible --
>
> (I) reproductive dysfunctions,

(II) neurological disorders,

(III) heritable genetic mutations, or

(IV) other chronic health effects.

() The chemical is known to cause or can reasonably be anticipated to cause, because of --

() its toxicity;

() its toxicity and persistence in the environment or,

() its toxicity and tendency to bioaccumulate in the environment, a significant adverse effect on the environment of sufficient seriousness, in the judgment of the Administrator, to warrant reporting under this section.

42 U.S.C. § 11023(d)(2). As Senator Stafford explained on the floor of the Senate during debate on the bill, this statutory standard is more relaxed than the usual standards used to regulate chemicals:

> The Administrator should recognize that he or she is not regulating a chemical by listing it under this subsection. There is no requirement to perform risk assessments or to balance the benefits and costs of a decision to list. The purpose of this section of the bill is to inform the public which can be accomplished without very great burden on the reporting facility. Therefore, a decision to list a chemical for the purposes of reporting under this section can and should be made on the basis of less scientific evidence than would be needed to regulate its manufacture, use or disposal (132 Cong. Rec. 14908 (Oct. 3, 1986)).

DINP very clearly meets the statutory standard. As discussed above, DINP is a known carcinogen and in addition has been shown to cause serious reproductive and developmental effects, as well as other toxic effects. The public has a right and a need to know who is producing and releasing DINP into the environment. DINP must be listed.

Conclusion

Because DINP is a carcinogen and reproductive and developmental toxicant, it must be listed as a Section 313 toxic chemical, under Congress's clear criteria. We urge you to add DINP to the toxic chemical list immediately.

Please direct all correspondence regarding this petition to Laurie Valeriano at 206-632-1545x14.

Sincerely,

Laurie Valeriano
Policy Director

REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological Profile for Di(2- ethylhexyl)phthalate (DEHP). U.S. Public Health Service (Apr. 1993).

Aristech Chemical Corporation (1995a). EPA/TSCA 8(e) Submission 8EHQ 0794-13083 (Jan. 13, 1995).

Aristech Chemical Corporation (1995b). EPA/TSCA 8(e) Submission 8EHQ 0794-13083 (Mar. 20, 1995).

Aristech Chemical Corporation (1995c). EPA/TSCA 8(e) Submission 8EHQ 0794-13083 (Apr. 12, 1995).

Babich, M., U.S. Consumer Product Safety Commission. "The Risk of Chronic Toxicity Associated with Exposure to Diisononyl Phthalate (DINP) in Children's Toys" (Dec. 1998).

Bio/dynamics Inc. "A Chronic Toxicity Carcinogenicity Feeding Study in Rats with Santicizer 900." Project Number 81-2572, EPA Document Number 86-870000362 (June 20, 1986) (with transmittal letter dated June 5, 1987).

Butala, J.H., *et al.* "Oncogenicity Study of Di(Isononyl) Phthalate in Rats." *Toxicologist* 30(1) pt.2:202 (Mar. 1996), Abstracts of the 35th Annual Meeting of the Society of Toxicology (Abstract No. 1030).

Butala, J.H., *et al.* "Oncogenicity Study of Di(Isononyl) Phthalate in Mice." *Toxicologist* 36(1) pt.2:173 (Mar. 1997), Abstracts of the 36th Annual Meeting of the Society of Toxicology (Abstract No. 879).

Gonzalez, F.J. (1997). "Recent Update on the PPAR_-Null Mouse." *Biochemie* 79:139-144.

Harris, C., *et al.* (1997). "The Estrogenic Activity of Phthalate Esters *In Vitro*." *Environ. Health Perspec.* 105(8):802-811.

Lee, B., U.S. Consumer Product Safety Commission. Memorandum regarding: "Health Effects Update, Acceptable Daily Oral Intake, and Carcinogenicity of Diisononyl Phthalate (DINP) (Aug. 31, 1998).

Lington, A.W., *et al.* (1997). "Chronic Toxicity and Carcinogenic Evaluation of Diisononyl Phthalate in Rats." *Fund. Appl. Toxicol.* 36(1):79-89.